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Chemical Warfare and the Gulf War: A Review of the Impact on Gulf Veterans' Health

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It is unlikely that Gulf War veterans are suffering chronic effects from illnesses caused by chemical warfare nerve agent exposure. Extensive investigation and review by several expert panels have determined that no evidence exists that chemical warfare nerve agents were used during the Gulf War. At no time before, during, or after the war was there confirmation of symptoms among anyone, military or civilian, caused by chemical warfare nerve agent exposure. However, studies of Gulf War veterans have found belief that chemical weapons were used, significantly associated with both severe and mild-moderate illnesses. The psychological impact of a chemical warfare attack, either actual or perceived, can result in immediate and long-term health consequences. The deployment or war-related health impact from life-threatening experiences of the Gulf War, including the perceived exposure to chemical warfare agents, should be considered as an important cause of morbidity among Gulf War veterans.

Introduction

The threat of a chemical war has a profound impact on all layers of society.[1] Prior to the Gulf War, Iraq had used chemical weapons on numerous occasions against Iran and its own citizens.[2] At the time of the deployment to the Arabian Gulf, the U.S. intelligence community had assessed that Iraq had a significant chemical weapons capability and had likely forward-deployed these weapons.[3,4] The initial objectives of the strategic air campaign were to (1) disrupt the Iraq leadership and command and control; (2) achieve air supremacy; (3) cut supply lines; (4) destroy Iraq's nuclear, biological, and chemical capability; and (5) destroy the Republican Guard. Destroying Scud missiles and mobile launchers became a priority early in the air campaign.[5] Although prepared to use them against coalition forces, the intelligence community has assessed that Iraq did not use chemical weapons during the Gulf War.[3]

Soon after the Gulf War, concern about potential environmental and biological and chemical warfare exposures arose stemming from controversy over the existence of a unique "Gulf War syndrome."[6] In 1994, the U.S. Senate Committee on Banking, Housing, and Urban Affairs issued a report expressing the belief that there was "reliable evidence that U.S. forces were exposed to chemical and possibly biological agents."[7] Following the public release of information on June 21, 1996, indicating that U.S. service members may have inadvertently been exposed to trace levels of chemical warfare nerve agents as a result of weapons destruction at Khamisiyah (an Iraq weapons depot), news reports appeared almost daily documenting this "probable" exposure and highlighting the lapses of Pentagon authorities in publicizing that information. However, throughout the U.S. occupation of Khamisiyah, including the demolition period, no reports were made of chemical warfare nerve agent detections, nor was there confirmation of symptoms consistent with chemical warfare nerve agent exposure.[8] Exhaustive investigation of other "exposure" incidents published in more than 60 individual reports has revealed similar negative findings.[9]

Individual investigators have reinforced unsubstantiated allegations that chemical warfare nerve agents were widely used in the Gulf War, for example, alleging, ". . . large numbers of military personnel were repetitively exposed to low environmental levels of the organophosphate chemical nerve agent sarin. . . ."[10,11]

Organophosphate Nerve Agents—Known Toxicology and Pathology

The toxicology, pathology, and medical presentation of acute and chronic health outcomes among individuals exposed to organophosphate (OP) pesticides and OP chemical warfare nerve agents have been the subject of extensive research over the last five decades. A number of reviews are available in the published literature and provide a strong basis for evaluation of health outcomes in humans exposed to OP pesticides and OP chemical warfare nerve agents (Table I).[12-33] Acute, limited exposures of brief duration with minimal to no clinical symptoms have not been shown to produce lasting health affects.[27,34,35] Subtle long-term neurological (primarily neurologic and psychiatric) effects may sometimes follow recovery from severe acute cholinergic poisoning and may last months or even years. These effects range from psychological, behavioral, or electrophysiological abnormalities to changes in the latencies of action potentials in skeletal muscles.[32,34,36-48] Some of these long-term effects may be mediated by cholinergic mechanisms, although other mechanisms have been hypothesized.[43] In survivors of the Tokyo subway sarin attack, the physical (cholinergic) signs and symptoms from the sarin intoxication disappeared within a few weeks in the vast majority of cases, although symptoms consistent with post-traumatic stress disorder and sleep disorders

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TABLE I

ACUTE AND CHRONIC OUTCOMES AMONG THOSE EXPOSED TO OP CHEMICAL WARFARE NERVE AGENTS

1. Acute cholinergic effects that occur minutes or hours following exposure, which may lead to mortality or recovery.
2. A delayed intermediate syndrome affecting muscles, expressed as muscle weakness, which can occur days following recovery from severe immediate acute effects and is reversible over days or weeks. (The intermediate syndrome is rarely seen in OP chemical warfare nerve agent exposure.)
3. A delayed peripheral polyneuropathy caused by a few OP pesticides usually occurring weeks following an acute exposure, variously called OPIDN, which is characterized by inhibition of the enzyme, neuropathy target esterase or neurotoxic esterase. (Only under unique experimental conditions will OP chemical warfare nerve agents induce delayed neurotoxicity.)
4. Subtle, long-term neurological (primarily neurologic and psychiatric) effects that sometimes follow recovery from severe acute cholinergic effects, which may last months or even years.

remained several months after the incident.[45] Genetic differences in activity levels of detoxification enzymes have been hypothesized to account for some individual variations in susceptibility, however this effect has not been observed clinically.[49]

A single high-dose or repeated low-dose exposure to some of these agents has been shown to produce a delayed peripheral polyneuropathy, referred to as OP-induced delayed neurotoxicity (OPIDN), which usually occurs days to weeks following exposure.[50] The OP chemical warfare nerve agents GA (tabun), GB (sarin), GD (soman), and VX, along with several close analogs, have been assessed for their ability to induce OPIDN and to inhibit neuropathy target esterase, thought to be the target for OP polyneuropathy.[51-54] In general, such neuropathies have been produced only at supralethal doses in animals kept alive by antidotal measures.

The scientific literature provides little evidence that subclinical exposure to sarin or cyclosarin, the chemical warfare nerve agents found at the Khamisiyah weapons depot, can result in long-term health consequences as a result of the agent's toxicity.[18-21, 29, 53, 55-59] In their review of Gulf War health issues, the Presidential Advisory Committee gave specific attention to the question of health effects of low-level exposure to chemical warfare nerve agents and concluded that the available scientific literature does not indicate that long-term, subtle, neuropsychological, and neurophysiological effects could occur in humans following low-level (asymptomatic) exposure. In the absence of exposure sufficient to produce observable acute health effects, there is no medical basis to infer that exposures to chemical warfare nerve agents, if such exposures occurred, were of sufficient magnitude to be responsible for any of the symptoms or health concerns among Gulf War veterans.

Health Outcomes among Gulf War Veterans—Potential Association with Chemical Warfare Nerve Agents

Numerous studies have examined patterns of morbidity and mortality among Gulf War veterans.[60-67] Epidemiological studies have found no increase in risk for postwar hospitalizations for various discharge diagnoses.[60-62,68] The postwar hospitalization experience of U.S. Gulf War veterans who were near Khamisiyah during nerve agent munition destruction was evaluated, looking specifically at several neurological outcomes that might be associated with chemical warfare nerve agent exposure. There was no indication that veterans potentially exposed to the nerve agent plumes experienced unusual postwar morbidity.[69] Neurological evaluations of referral patients participating in the Department of Defense (DoD) Comprehensive Clinical Evaluation Program found no consistent patterns of neurologic disease or new neurologic syndromes.[70] In a study looking at 7-year postwar mortality, Kang and Bullman[67] found the excess of deaths among Gulf War veterans attributed to motor vehicle accidents that was observed during the earlier postwar years had decreased steadily to levels found among non-Gulf veterans and that the overall risk of death for both Gulf War veterans and non-Gulf War veterans was less than one-half that expected in their civilian counterparts. The risk of death from natural causes remained lower among Gulf War veterans compared with non-Gulf War veterans, and there was no statistically significant difference in cause-specific mortality among Gulf War veterans relative to potential nerve gas exposure. Epidemiological studies of varied groups of American, British, and Canadian Gulf War veterans have found that subjective symptoms are reported more frequently among those who served in the Gulf War than Gulf War-era personnel who were not stationed in the Gulf.[60,63,71-76] Dlugosz et al.[77] found that Gulf War service was associated with greater risk for postwar hospitalization for acute reactions to stress and that personnel who served in the ground war were at greater risk for postwar drug-related disorders and alcohol-related disorders.

Nisenbaum et al.[78] and Spencer et al.[79] examined the association between self-reported stressors, including perception of a chemical warfare agent threat (belief that chemical weapons were being used), experienced during the Gulf War deployment and a postulated chronic multisymptom illness. These investigators found belief that chemical weapons were being used, in addition to various other stressors, were significantly associated with both severe and mild-moderate illnesses using univariate and multivariate analysis. These results are consistent with results from other studies that examined the health impact of a perceived threat of chemical weapons.[73,80] Such stressors may cause or exacerbate illness because of the psychological and physiological maladaptive stress response.[78] These findings do not support the hypothesis of Haley and Kurt [81] that unexplained illness among Gulf War veterans is a result of neurotoxic effects caused by exposures to chemicals that inhibit cholinesterase activity.[78,79]

Several published studies have examined associations between symptoms and self-reported exposures to a wide range of hazards.[73,82,83] As early as 1993, preliminary findings suggested that a high percentage of Gulf War veterans experienced some stress-related symptoms after returning to the United States.[84,85] Conclusions from the 1994 National Institutes of Health Technology Assessment Workshop indicated that stress from the imminent threat of biological or chemical warfare may

be a common thread linking some Gulf War veteran's symptoms.[85] The self-reported exposures to toxicants or hazards have been significantly related to virtually all of the health outcomes studied. The associations between multiple unrelated exposures and multiple unrelated symptoms are difficult to explain from a toxicological perspective.[60,87,88]

Other studies have searched for relationships between self-reported symptoms, exposures to combat, poisonous gas, or occupational exposure to petroleum products, and measures of stress.[60,75,89-92] Associations between war-related stress and physical symptoms of ill health were found, but these studies do not indicate the strength of the relationship and do not exclude possible relationships between symptoms and other risk factors.[89-91]

Proctor et al.[75], using a priori hypotheses about the toxicant effects of exposure to specific toxicants, examined the relationships between self-reported exposures and body-system symptom groupings through multiple regression analyses, controlling for war zone exposure and post-traumatic stress disorder. Self-reported exposures to pesticides, debris from Scuds (Iraq's short range ballistic missiles), chemical and biological warfare agents, and smoke from tent heaters were each significantly related to increased reporting of specific predicted body-system symptom score groupings. Using the nerve agent pretreatment pyridostigmine bromide as a correlate for chemical warfare threat, the findings of Schumm et al.[93] would support increased long-term declining subjective health status among female reserve component Gulf War veterans most exposed to the perceived threat of chemical warfare nerve agents.

Most studies have found that self-reported exposures are significantly correlated to many of the medical and psychiatric conditions, suggesting that no single exposure was related to the outcomes observed and that health complaints of Gulf War veterans are not consistent with a unique syndrome.[75,76,92,94,95]

Several studies that reported associations between self-reported chemical warfare nerve agent exposures and health effects overlooked the effects of exposure misclassification error on their results.[96] Demonstration of the difference in self-reported exposures and outcomes among various groups of Gulf War veterans has been observed in various studies.[60,81,96-98] Gulf War veterans studied by Haley et al.[81] and Gray et al.,[60] many of whom slept and worked in the same areas, reported very different wartime exposures, especially chemical and other environmental exposures. Studies that make sole use of self-reported findings, for both outcome and exposure, must be viewed with caution.[97] Observational data are always subject to potential biases and study results must be independently replicated. One explanation for the findings of Haley et al.[81] is information bias, that is, persons who report symptoms are also more likely to report exposures.[99]

Several studies have included neurophysiological and neuropsychological evaluations of symptomatic Gulf War veterans.[75,100-111] In general, these studies have not found obvious, consistent, or specific changes in objective measures of numerous neurophysiological or neuropsychological variables. Several hypotheses concerning the cause or physiological basis of difficult-to-diagnose chronic illnesses among some Gulf War veterans remain unconfirmed. Some investigators hypothesize relationships to stress, whereas other investigators hypothesize relationships to low-level chemical warfare nerve agent exposure and yet other studies have not revealed neurological signs or symptoms that would support these hypotheses.[81,96,100,103,112-117]

Jamal et al.[105] compared symptomatic Gulf War veterans with a matched group of "healthy" civilian controls. Although significant differences in three measures of peripheral nerve function were observed, the authors were cautious in their conclusions. The finding that symptomatic subjects, from any group, have differences in health compared with asymptomatic subjects is not surprising.

Using an exploratory, mathematical data structure technique, Haley et al.[98] identified and named six possible syndromes based on clustering of symptoms. The investigators intensely studied a small number of the most symptomatic subjects with the three syndromes showing the strongest associations among symptoms, using an extensive multidisciplinary testing protocol. Haley and Kurt[81] hypothesized that these factor analysis-derived syndromes may represent variants of OPIDN due to exposure to mixtures of anticholinesterase agents (e.g., chemical warfare nerve agents, pesticides, insect repellent, and/or pyridostigmine bromide). However, such "OPIDN variants" have not been previously reported, and the suggestion that veterans of the Gulf War have a form of OPIDN is "without support."[93,118] The study design flaws and interpretation of the Haley et al.[81] findings have been discussed elsewhere.[12,97,99,119-125] The relevance of these findings remains to be shown and other investigators have not been able to replicate these results using larger, more representative samples.[73,74,76,95,126]

The Psychological Consequences of Chemical Warfare

The psychological trauma of experiencing a potential or real attack from chemical warfare agents along with the psychological and psychosocial consequences of combat and deployment presents a confounding factor in evaluating health effects of actual exposures.[127,128] The threat or perceived exposure to chemical warfare agents has been shown to have a lasting and adverse impact on human health.[15,44,129,130] Some veterans of the Gulf War believe unequivocally that they were exposed to chemical warfare nerve agents.[86] Extensive monitoring capabilities for chemical warfare agents were deployed to the Arabian Gulf and the threat from Iraq's chemical warfare weapons was perceived as a real and present danger.[3,131] Gulf War veterans frequently heard sirens and other warnings issued as a result of possible agent detection from chemical warfare detection devices and spent many hours waiting in cumbersome protective gear.[86] Often, troops were not given a full explanation for the cause of the alarm or informed that the alarm was a false alarm. Troops generally were unaware of the extent that chemical warfare agent monitoring equipment could register false alarms, leading to a belief that they had been subject to attack by chemical warfare agents, when in fact they had not.[132]

Similar to the experience of troops during World War I, the dread of chemical warfare, once aroused, was sustained and disseminated by the precautions taken against the agent rather than by exposure to it.[86,132,133] A quote from L. F. Haber, *The Poisonous Cloud: Chemical Warfare in the First World War*, typifies the impact on health that the belief a chemical warfare attack occurred can have: "It is possible that these men, in their maturity, would attribute their condition to the consequences of

a gas attack ten or fifteen years earlier. . . , but it was enough for people to believe that being gassed was the cause of illness years later, and thus the special anxiety created by chemical warfare continued into the peace and was kept alive in the public's consciousness."[133]

A chemical or biological attack is a potent form of psychological warfare, whether that attack is real or a cleverly designed hoax and whether a lone sociopath initiates it, a group of domestic or foreign terrorists, or a rogue nation.[134] Military operations in a potential chemical environment are associated with intense fear of that form of warfare. Ursano[135] attributes fear among military personnel to (1) the particular, personal psychological fears of chemical warfare agents, (2) a sense of the need to continue to operate after a chemical warfare attack, and (3) the indiscriminate nature of chemical warfare agents. The feelings of helplessness in the face of a ubiquitous and unseen killer can be overwhelming.[23,136-138] The psychological impact of a potential or actual chemical attack can result in immediate causalities from acute stress disorder, grief, anger, scapegoating, and somatization disorders.[134] Longer term effects include phobias, sleep disorders, post-traumatic stress disorder, substance abuse, and major depression.[139,140]

These findings are consistent with observations from military training using mock warfare conditions and from civilians responding to the belief that they are under attack with chemical warfare nerve agents.[139-143] In a study involving 366 military personnel involved in training exercises, the chemical and biological warfare combat environment itself was responsible for 5% to 20% of causalities even in the complete absence of exposures to chemical and biological warfare agents.[143] Investigators found 10% to 20% of participants experienced symptoms, including anxiety, claustrophobia, and panic. During the Gulf War, a number of military members presented with panic, hyperventilation, and inability to wear respirators when chemical alarms were sounded, despite a perceived real chemical threat.[144-146]

As with military personnel, civilians who believe that they are under attack from chemical or biological weapons also experience the physiological manifestations of severe stress reactions.[138] During the Gulf War, Israel was subjected to attacks by Scud missiles, several of which fell in highly populated areas causing human injuries and extensive property damage.[147] Although it was confirmed that none of the Scuds were armed with chemical warfare agents, the uncertainty in time, place, and type of warhead, conventional or chemical, was a source of chronic stress and the immediate cause for many traumatic stress reactions at or near the missile attack sites.[148] Somatic reactions were more prevalent among women than men and among those who had only completed elementary school.[148]

Conclusion and Future Research Needs

Extensive investigation and review by several expert panels have determined that no evidence exists that chemical warfare nerve agents were used during the Gulf War. At no time before, during, or after the war was there confirmation of symptoms, among military or civilian personnel, caused by chemical warfare nerve agent exposure. However, the findings of U.N. inspections and investigations following the Gulf War indicate that some U.S. troops potentially may have been exposed to trace levels of sarin and cyclosarin.[5,149]

Given the limited extent of exposure, what is the likelihood that chemical warfare nerve agents are a cause of long-term health problems among Gulf War veterans? Chemical warfare nerve agent exposure is a very unlikely cause of the postulated "Gulf War syndrome" or any illness among Gulf War veterans. Reviews of the literature indicate that long-term health effects from exposures do not occur unless there are symptoms immediately following exposure.[12, 18-23, 28, 32, 57, 150-152]

There is no medical or scientific evidence for an atypical OPIDN following asymptomatic exposure to chemical warfare nerve agents.[12] Systematic examinations of over 100,000 Gulf War veterans have failed to identify any pattern of neurological damage that would indicate harm from exposure to chemical warfare nerve agents.[60-69]

Consistent scientific evidence indicates that service in the Gulf War resulted in increased reporting of symptoms among Gulf War veterans.[60,63,73-76] Neither chemical exposure data nor toxicological evidence from the scientific literature offer potential explanation for the observed symptoms.[79] One consistent finding has been the recognition of psychological conditions and related symptoms as a major diagnostic category among Gulf War veterans.[153] This finding is not unique to Gulf War veterans, as similar symptoms have been observed among veterans of many wars of the past century.[154-156] Physical and mental stress associated with combat manifests in diverse ways and is likely to be an important contributing factor to the broad range of physiological and psychological illnesses among military forces.[154,157]

It is unlikely that humans will ever again be deliberately exposed in a research setting to chemical warfare nerve agents. Therefore, any new human data on long-term effects will have to come from study of people exposed years ago, of individuals exposed to chemical warfare nerve agents accidentally, through acts of terrorism, or war, or individuals exposed to related agents such as OP pesticides. Detailed toxicology studies on the long-term effects of low-level exposures will, by necessity, use animal models and will have to be appropriately designed to facilitate animal-human extrapolations. There are limitations as to the ability of the experimental animal studies to adequately model the subtle neuropsychological deficits, if any, which would follow exposures to subclinical levels of chemical warfare nerve agents. It is important that the exposures used in these studies be similar in route of administration, intensity, and duration as those experienced by troops under actual deployment conditions.

Although it is unlikely that Gulf War veterans are suffering chronic effects from illnesses caused by chemical warfare agent exposure, further research is ongoing to determine whether there are synergistic effects among varied agents, including psychological stressors, pesticides, and pyridostigmine bromide, a pretreatment for chemical agent exposure.[87,158] Further research also is ongoing to develop more sensitive and specific chemical warfare detectors, both to provide early warning and to better document exposure for later assessment of health effects. The current recognized biomarker of exposure to OP chemical warfare nerve agents, inhibition of red blood cell acetylcholinesterase, has limitations when applied to retrospective analysis.

as well as being a poor index of toxicity. New techniques are emerging from ongoing research that may assist in retrospective analysis of exposure.[159] As part of the federal response to the Khamisiyah announcement, a number of experimental animal studies examining the neurologic and neurophysiologic sequelae of low-level exposure to sarin were begun. These studies, generally undertaken in 1998, have not yet produced a corpus of data published in the peer-reviewed literature. The annually updated summaries of progress in this research area can be found on the Internet (http://www.va.gov/resdev/prt/gulf_war_2000/). The DoD has developed a strategy to address low-level exposures to chemical warfare nerve agents.[160] The proposed DoD strategy and supporting 5-year research plan are designed to yield data that will guide the potential evolution of policy and doctrine. The DoD must improve upon existing capabilities for protective and responsive measures against chemical warfare agents. Training on the limitations of chemical agent detectors and documentation and communication at all levels on false alarms from detectors should be an important part of this improved policy and doctrine.

In an effort to respond to the health concerns of Gulf War veterans and their families, the Department of Veterans Affairs contracted with the Institute of Medicine (IOM) to study the scientific evidence concerning associations between agents to which Gulf War veterans may have been exposed and resulting adverse health effects.[59] For sarin, the IOM Committee concluded that (1) there is sufficient scientific evidence of a causal relationship between exposure to sarin and a dose-dependant acute cholinergic syndrome that is evident seconds to hours subsequent to a sarin exposure and resolves in days to months; (2) there is limited/suggestive evidence of an association to exposure to sarin at doses sufficient to cause acute cholinergic signs and symptoms and subsequent long-term health effects; and (3) there is inadequate/insufficient evidence to determine whether an association does or does not exist between exposure to sarin at low doses insufficient to cause acute cholinergic signs and symptoms and subsequent long-term adverse health effects. Although the IOM Committee recognized the impact on health from the threat of or perceived exposure to chemical and biological warfare agents, the committee failed to examine the potential association between this significant exposure and ill health among Gulf War veterans.

Psychological conditions have been consistently recognized as a major diagnostic category among Gulf War veterans. These conditions have been associated with the perceived exposure to chemical warfare agents. The Department of Veterans Affairs and IOM should examine the health impact from life-threatening experiences during the Gulf War, including the perceived exposure to chemical warfare agents, as an important health concern and cause of morbidity among Gulf War veterans.

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14. ABSTRACT (maximum 200 words)
It is unlikely that Gulf War (GW) Veterans are suffering chronic effects from illnesses caused by chemical warfare (CW) nerve agent exposure. Extensive investigation and review by several expert panels have determined no evidence exists that CW nerve agents were used during the Gulf War. At no time before, during, or after the war was there confirmation of symptoms among anyone, military or civilian, caused by CW nerve agent exposure. Studies of GW veterans have found belief that chemical weapons were used, significantly associated with both severe and mild-moderate illnesses. The psychological impact of a CW attack, either actual or perceived, can result in immediate and long-term health consequences. The deployment or war-related health impact from life threatening experiences of the GW, including the perceived exposure to CW agents, should be considered as an important cause of morbidity among GW veterans.

14. SUBJECT TERMS
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